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THE RELATION OF ORGANIC DUST TO PNEUMONOKONIOSIS*

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TWO and a quarter centuries ago, Ramazzini published his work on the ill effects of the inhalation of dust. He believed that all dusts possessed the power of producing changes in the respiratory tract. From that time until comparatively recently the belief that both organic and inorganic dust could produce pneumonokoniosis has been accepted. In addition to this traditional belief, several other factors have had a great influence, especially the association of tuberculosis with dust infections. From the very beginning it was noted that workers exposed to dust often developed tuberculosis. The belief that this was true of all forms of dust was at one time quite generally accepted. Recently there has been a tendency to restrict this predisposition to tuberculosis to certain of the inorganic dusts. Not only that, but it is asserted that the inhalation of some of the inorganic materials, notably coal dust, is a protection against tuberculosis. This latter statement is, I believe, still debatable. At any rate, it is certainly true that quite a large proportion of the workers suffering from anthracosis do develop tuberculosis; whether the coal dust prevents still more from developing the disease is something yet to be proved.

Assuming that the inhalation of the inorganic dusts, notably silica, was attended with a high incidence of tuberculosis, it was relatively easy to extend this dictum to include all forms of dust, no matter what their character might be. Probably the most potent influence in recent years in establishing this belief was the work of Hoffman. This observer published statis-

*Read by title at the meeting of the American Association of Physicians, May, 1924. Received for publication Oct. 23, 1924.

tics based on the experience of the Prudential Insurance Company, which seemed to show that the tuberculosis death rate was uniformly high in all industries in which dust was a factor. He made no distinction as to the type of dust, nor did he attempt to show the effect of other influences. The fallacy of Hoffman's deductions has been exposed by Winslow and Greenburg.[1] These writers point out that Hoffman used as a norm for comparison the ratio for males in the registration area obtained from the data of the United States Census Bureau. On this basis almost all the dusty trades which he tabulates show a surprisingly high tuberculosis ratio. Winslow and Greenburg believe that the comparison between the Prudential figures for a given dusty trade and the census figures for all occupied males is not a fair one, and that the conclusion indicated by such a comparison is unwarranted. The point that I wish to make is that the assumption that all dusts are injurious to the lungs is unwarranted, and, so far as the organic dusts are concerned, there is no pathologic proof that they are *per se* harmful. It has been assumed that they are injurious simply because certain groups of workers exposed to organic dust show a high incidence of tuberculosis. As Winslow and Greenburg point out, it is essential that the effect of industrial hazards shall not be complicated by the influence of social and economic factors of a more general nature.

Another element that has added to the confusion is the disregarding of the fact that the dust may be mixed in character. Thus, while the industry may be ostensibly one in which organic material is causing the damage, analysis of the dust itself will show a high inorganic content, and it is the latter which is producing the pneumonokoniosis. Many years ago, Greenhow incinerated the lungs obtained from two flax dressers. In both instances the resulting ash contained a large amount of silica. In other words, the workers, while apparently exposed to organic dust, were really inhaling inorganic material, and it was the inorganic element that caused the pneumonokoniosis. Pancoast and I have had the same experience in a roentgenologic study of a group of carpet weavers. In this instance, the roentgenograms showed changes differing from those in other groups exposed to organic material. An analysis of the dust in this factory showed that it had a large amount of inorganic material. Nor is it safe to assume that because workers handle certain organic materials any disease they may develop is of necessity due to the inhalation of dust from that material. A notable instance of this is the cigar-making trade. The incidence of tuberculosis is high in this industry, and some have attempted to show that it is due to the inhalation of powdered tobacco, which irritates the lungs and predisposes the individual to tuberculosis. As I have shown elsewhere,[2] there is no dust in this occupation. The tobacco from the time it is received by the manu-

[1] Winslow, C.-E. A., and Greenburg, L.: Industrial Tuberculosis and the Control of the Factory Dust Problem. THIS JOUR., 1920–1921, *2*, 336.

[2] Landis, H. R. M.: The Pathological and Clinical Manifestations Following the Inhalation of Dust. THIS JOUR., 1919–1920, *1*, 117.

facturer until it is made into cigars is in a moist, pliable condition. Furthermore, all of the tobacco leaf not used in the cigar is carefully saved and used for other purposes. This accounts for the entire absence of tobacco dust.

Probably the most striking feature in connection with the dust problem is the failure to bring forth the proof that organic dusts do produce pathologic changes. The usual procedure is to study the morbidity and mortality rates of a given dusty occupation, and then to assume that any respiratory illness that may be encountered is to be ascribed to the inhalation of the dust. The proof should be easily produced (1) by the postmortem examination of workers so exposed, and (2) by the roentgenologic study of such workers. In the case of inorganic dust both of these tests have been applied. There is ample evidence at hand to show that inorganic dust, if inhaled over a sufficiently long period, will produce just as definite and characteristic lesions as do any of the bacterial infections. A man who works in a coal mine or in a pottery sufficiently long will inevitably develop a pneumonokoniosis. The changes may occur more quickly and may be more extensive in one person than in another, even if the conditions are identical, but there is no question as to the ultimate result. Furthermore, the dust itself has been recovered from the lungs. In fact, the progress of the pulmonary changes is so definite that, given the type of dust and the length of exposure, one can almost predicate the roentgenologic findings.

Exactly the reverse is true in the case of workers exposed to organic dust. Nowhere in all that has been written on the subject of pneumonokoniosis has there been brought forward any pathologic evidence to show that any of the organic materials create changes similar to those produced by inorganic dust, nor have any of the common organic materials, such as cotton, silk, wool, hemp, hair, etc., ever been demonstrated in the lungs. As far as the X-ray evidence is concerned, the subject can be dismissed with the statement that individuals exposed to organic dust show no more marked changes than are encountered in those who have dwelt all their lives in large cities. When the evidence of pneumonokoniosis is slightly more marked than normal in these cases, it is to be ascribed to the fact that there is a considerable amount of inorganic material mixed with the organic dust.

During the past year, I have been able to obtain complete autopsy protocols on fifty persons who had been employed for varying lengths of time as textile workers (weavers, spinners, knitters, etc.), occupations having to do with the handling of cotton, silk, and wool. In many of the mills the cotton fiber hangs like a cloud in the workroom. There can be no doubt that fibers exist in sufficient quantity and of sufficient fineness to reach the lungs and produce changes, if that material were capable of doing so. Furthermore, in view of the typical changes noted in the lungs of workers exposed to inorganic dust, one would expect the description of the lungs of organic dust workers to show recognizable changes. The fifty autopsies were obtained with some difficulty from three of the large hospitals in

Philadelphia. Although there are admitted to the hospitals many patients giving their occupations as textile workers, the number available who have been employed over a sufficiently long period of time, and who die and are autopsied, reduced the available material very markedly. Admitting that the number is small, it is interesting to note that in no instance was there the slightest reference to changes in the lungs comparable to those noted in the case of those exposed to inorganic dust. In all cases the trade had been the only one followed. The ages of the workers as shown in the following tabulation indicate that nearly all were beyond the middle period of life, and that a large proportion were of advanced years.

Age Group	*Number*
30–39	5
40–49	15
50–59	12
60–69	12
70 and over	6

In the pottery industry, with which I am most familiar, the damaging effects of the inorganic dust become more and more noticeable after the age of 40. There are relatively few workers remaining at the trade beyond the age of 60. They become incapacitated as the result either of a pneumonokoniosis or of a superimposed tuberculous infection.

The causes of death in the group of workers exposed to organic dust were as follows:

Carcinoma	10
Cardiovascular disease	10
Renal disease	7
Apoplexy	4
Lobar pneumonia	4
Tuberculosis, pulmonary	4
Organic nervous diseases	4
Intestinal obstruction	1
Laryngeal stenosis	1
Typhoid fever	1
Erysipelas	1
Alcoholism	1
Gas poisoning	1
Edema of lungs	1

The terminal infection as shown by the autopsy was as follows:

Terminal bronchopneumonia	7
Tuberculosis	5
Lobar pneumonia	4
Edema and congestion of lungs	1
Carcinoma of lungs, metastasis	1

The causes of death in this group of cases are in marked contrast to the usual death returns in the case of inorganic dust workers. Among the latter, death is in the great majority of instances ascribed to some respiratory condition. While the actual cause of death is rarely, if ever, given as pneumonokoniosis, but rather as some secondary factor, such as bronchitis, pleurisy, emphysema, bronchiectasis, tuberculosis, etc., it serves to indicate that the predominant clinical features of the illness were respiratory in character.

Among the group under consideration the causes of death are rather what one would expect in the ordinary run of cases falling in these age periods. The only possible exception is that of tuberculosis. It is quite possible that with a larger number of cases the percentage from this cause would be less. The object of the present paper, however, is not to determine whether organic dust acts either as a carrier of the tubercle bacillus or as a predisposing cause of tuberculosis, but to point out that it has no power to establish the condition known as pneumonokoniosis. In the first place, the roent-

genograms of the chest show no changes analogous to those so readily demonstrable in the case of inorganic dust. In the second place, there is no pathologic proof based on autopsy experience that such is true. Finally, it is essential for practical purposes to keep the effects of inorganic dust and of organic dust separate. The former presents a definite problem of prevention. Disregarding the predisposing influence it may have on the development of tuberculosis, it should be clearly recognized that inorganic dust of itself can and does cause serious and disabling damage to the lungs. In every group of workers so disabled a considerable proportion of them are suffering from a pure pneumonokoniosis, without the added tuberculous infection.

Organic dust, on the other hand, judging from the evidence at present available, never produces the condition known as pneumonokoniosis. That some organic dusts give rise to protein intoxication, such as asthma, threshers' fever, etc., is known. Further studies may extend the list and at the same time may demonstrate the fact that this type of dust may be a source of danger in acting as the carrier of bacteria into the respiratory tract.

INDUSTRIAL POISONING BY ARSENIURETTED HYDROGEN (ARSINE)*

DR. I. GUELMAN

Assistant Director of the V. A. Obuch Institute of Moscow for the Investigation of Occupational Diseases

ARSENIURETTED hydrogen, or arsine, is one of the poisons met with from time to time in industry. It is generated, although not in very considerable quantities, during many processes of chemical and metallurgical industries. Among the kinds of work in which this highly dangerous and volatile gas may be generated, Glaister (1) includes the following:

Chemical analysis and laboratory work.
Production of aniline dyes.
Smelting of zinc polluted with arsenic.
Smelting and obtaining mineral ores.
Production of iron sulphate and several other chemical substances.
Inflating aerostats with hydrogen.
Soldering and galvanizing.

Arsine is a colorless gas, with a garlic-like smell, which passes into a liquid state at a temperature of −40°C. It is soluble in water in the proportion of about 5 volumes to 1 volume of water. Its production is accompanied by a considerable absorption of heat, which renders it highly unstable. It is most frequently generated during the treatment of zinc, tin, and iron with sulphuric and hydrochloric acids, because these substances are used in a crude, impure state and contain greater or lesser quantities of arsenic. Thus the sulphuric acid used in industry may contain from 0.045 to 0.140 per cent. of arsenious acid, and hydrochloric acid may contain from 0.0014 to 0.691 per cent. The zinc, iron, and tin, also, may contain considerable quantities of arsenic.

The reaction for the formation of arsine is expressed in the following formula:

$$H_2SO_4 + Zn = ZnSO_4 + H_2$$

The arsenic in acids and metals may then combine with this nascent hydrogen, forming arsine.

Arsine is highly poisonous. As to the amount which is dangerous, "Koelsch, basing his view on experiments on animals by Dubitzki, considers 0.3 gram AsH_3 (equivalent to 86.1 cc.) as a fatal dose for an adult man, or less—0.1 to 0.15 gram AsH_3—if the haemolytic action of the gas on different kinds of blood is made the basis. An actual instance where death followed inhalation of 0.6128 gram (equivalent to 175 cc.) AsH_3 suggests a much larger amount as the lethal dose, and Wignall, from analyses of the amount of arsenic excreted in the urine of workmen suffering from AsH_3 poisoning, comes to the same conclu-

*Received for publication May 5, 1924.

sion. Dubitzki (quoted by Koelsch) considers that a concentration of 3 parts per 100,000 would cause symptoms in time" (2). The concentration of arseniuretted hydrogen in the air rarely, however, attains such a height—a fact which may partly be accounted for by the instability of the gas. For this reason, the number of cases of acute poisoning elicited by medical registration is far from considerable.

In 1908 Glaister succeeded in finding in the entire returns at his disposal only 120 cases of poisoning due to arsine (1, pp. 100, 252). Koelsch (3) in 1920 summarized 130 alleged cases, thirty-six (27.7 per cent.) of which resulted in death. In England from 1900 to 1919 fifty-three cases of poisoning were registered (2, p. 307), the branches of the respective industries being as follows:

Chemical industry	28
Galvanizing works	7
Bronzing of art metal	5
Dyeing processes	4
Others	9

A very interesting instance of general poisoning is one reported by Dudley (4), in which thirty men belonging to the crew of a submarine were involved. The poisoning was due to the corroding of the lead plates in the batteries, which contained 0.2 per cent. of arsenic.

These figures do not, of course, include the total number of cases of poisoning from arsine. Under the present system of industrial poisoning registration, it is only the most acute and serious cases that are returned; cases of less acute and chronic poisoning, which are the type most frequently met with, evade registration.

To this comparatively small number of cases of acute arsine poisoning, we may add twelve new cases treated by the Moscow Institute for Investigation of Occupational Diseases. On September 23, 1923, eight patients were brought to the clinic of the Institute with signs of acute poisoning. Questioning the patients elicited the information that they had all been working in the galvanizing and etching department of a zinc plant until 2 o'clock on the previous day (one had worked until 4 o'clock) and that they had all been taken ill after a period varying from two to ten hours after the work. On the day in question thirteen men had been at work, of whom two were etchers and two galvanizers; the rest were laborers, who carried articles from the etching tank to the galvanizing tank. Eight of the thirteen were admitted to the clinic on September 23, and four with symptoms of less acute poisoning were suspended by us from work and sent to the clinic. The thirteenth man was in good health and did not go to the clinic.

The source of poisoning was an etching tank with a capacity of 200 poods[1] into which there had been poured the day before a bottle of sulphuric acid described by the workers as "suspicious-looking." In this tank on September 22, at about 12 o'clock, damaged galvanized vessels were immersed for a second treatment. During both the first and the second immersion of the vessels a white gas, similar in odor to hydrogen sulphide, was given off. All the men engaged in this work were taken ill with signs

[1] One pood = 36.113 pounds, or 16.38 kg.

of acute poisoning, one of them an hour or two after work. One of the workers, an etcher engaged in immersing vessels in the tank and detained at the work longer than the rest, felt ill while at work and exhibited signs of very acute poisoning. He died on the fifth day.

A detailed chemical investigation showed the causes of the poisoning; but the clinic had forestalled the results of the chemical analysis and, basing its conclusion on the symptoms and course of the disease, had established the fact of poisoning with arsine. The symptoms of poisoning were curiously similar in all the cases, differing only in degree. To make clear the symptoms of poisoning, let us summarize the course of three cases of acute and two cases of slight poisoning.

CASE 1.—X, aged 36, an etcher, who worked at the filling of the tank longer than any of the others (about four hours), was taken ill while at work, with headache, general weakness, and vomiting.

Physical Examination.—The patient on examination was found to be suffering from acute griping pains in the abdomen, especially in the region of the stomach and liver; from tenderness in the region of the kidneys; and from thirst. Physical examination elicited the following features and symptoms: drowsiness; general cyanosis of a copperish-red color; blurred and diffusely hemorrhagic conjunctivae; pupils narrowed and reacting very slightly; a scarlet, coated tongue; hiccuping and frequent vomiting of a large quantity of a dark greenish tar-like liquid (bile). The abdomen was sunken; the liver enlarged, projecting a finger's breadth from under the edge of the ribs; and the gallbladder palpable. The pulse was 90 in rate, weak and regular; the heart sounds were muffled. The respiratory rate was 24 per minute, and a few dry râles were audible. Tendon reflexes were heightened and there was clonus of the foot. The fecal material was darker than usual.

On the evening of the following day there was hiccuping, incessant vomiting, and continued thirst. The pupils were narrowed and the patient's color assumed a more jaundiced character. His temperature rose on September 24 and remained at the level of 37.7° to 38.1°C. until the day of death, when it fell to 35.6°.

During the course of the disease phenomena of acute oliguria were noted (20 to 30 c.c. of urine per day), together with hemoglobinuria, hiccuping, incessant vomiting, pain along the muscles and nerves, heightened reflexes, and jaundice increasing for two days and then decreasing.

Laboratory Findings.—Interesting phenomena were observed in connection with the urine and the blood. The examination of the urine, obtained by means of a catheter on September 23, gave the following results: day quantity, 50 to 60 c.c.; consistency, thick; color, brownish red; reaction, slightly acid; specific gravity, 1.019; albumin, 4.5 per cent.; Gmelin's reaction, negative; one to two kidney epithelial cells and three to four erythrocytes in each field. On September 26, the day quantity was 20 c.c.; the color, cherry red; albumin 5 per cent.; Gmelin's reaction, negative. A microscopic examination showed thirty to forty erythrocytes, about thirty leukocytes and one to two granular casts in each field.

Examination of the blood, which unfortunately was made but once, showed on September 24: erythrocytes, 3,020,000; hemoglobin, 48 per cent.; color index, 0.79; and leukocytes, 30,000. A differential count showed: neutrophils, 80 per cent.; basophils, 1 to 2 per cent.; lymphocytes, 12 per cent.; mononuclears and transitionals, 5 per cent. The proportion of leukocytes to erythrocytes was 1 to 100. Basophilic granulation and polychromatophilia of the red cells were noted.

Death followed on September 27 from failure of the heart.

CASE 2.—Y, aged 18, who was employed in receiving vessels from the galvanizing tank, had been working on the day of the poisoning from 8 A.M. till 1 P.M. He was taken ill at 3 P.M. with headache, weakness, and pains. At 6 P.M. an incessant vomiting

with bile in the vomitus began and continued throughout the whole evening and night. The vomiting was accompanied by pains in the abdomen. The patient turned yellow in the course of a few hours.

Physical Examination.—An investigation made at the Institute on the evening of the following day showed the patient to be semiconscious and suffering from giddiness, slight headache, violent thirst, nausea, incessant vomiting of dark, blackish-green bile in large quantities, from pains in the abdomen, especially in the upper part and about the liver, and from pain along the course of the nerves in the upper and lower extremities. The patient showed a marked jaundiced coloring of the cutaneous and mucous surfaces. His pupils were somewhat narrowed and the pupil and tendon reflexes sluggish. The liver was somewhat enlarged (2 to 3 cm. below the edge of the ribs); the spleen could not be palpated. Heart tones were dull and a few dry râles could be detected in the lungs. Respiration was 20; pulse, 80 to 100, regular and medium; blood pressure, systolic 115, diastolic 60. The stools were obstructed but of normal color.

Laboratory Findings.—Examination of the urine on September 24 showed: color, dark brownish red; specific gravity, 1.017; reaction, slightly acid; albumin, 4.5 per cent.; Gmelin's reaction, negative; three to five epithelial cells and two to three erythrocytes in each field, and crystals of hematoidin in the sediment. On September 26, the urine was slightly orange colored; specific gravity, 1.015; albumin, 0.66 per cent.; Gmelin's reaction, negative. Two to three leukocytes were present in each field; the sediment also showed epithelial cells containing small, brown needle-like crystals of hematoidin (bilirubin?).

Examination of the blood showed on September 24: erythrocytes, 2,137,000; hemoglobin, 37 per cent.; color coefficient, 0.89; leukocytes, 20,400 (differential count: neutrophils, 76 per cent.; eosinophils, 1 per cent.; lymphocytes, 20 per cent.; mononuclears, 4 per cent.); proportion of leukocytes to erythrocytes, 1 to 106. Examination of the erythrocytes showed basophilic granulation, polychromatophilia, and anisocytosis.

On September 28 the findings were: erythrocytes, 1,500,000; hemoglobin, 25 per cent.; color index, 0.83; leukocytes, 18,000 (neutrophils, 63 per cent.; basophils, 11 per cent.; lymphocytes, 12 per cent.; mononuclears, 14 per cent.); proportion of leukocytes to erythrocytes, 1 to 84.

On October 3, the hemoglobin figure was 56 per cent. Basophilic granulation, anisocytosis, polychromatophilia, and an increased number of thromboeytes were observed during the examination of the red cells.

Characteristics of the further course of the disease were a rapid disappearance of hemoglobinuria, albuminuria, and jaundice, with a slow regeneration of the blood. After leaving the hospital, the patient complained of pains in the hands and along the course of the nerves.

Case 3.—Z, aged 21, an etcher, worked in the etching department till 2 o'clock. After three hours' work he developed weakness, pains in the abdomen, nausea, vomiting of bile, and bloody urine.

Physical Examination.—An examination at the Institute showed the patient to be fully conscious but suffering from headache and giddiness, from nausea and frequent vomiting of thick, tar-like bile, from acute pain in the regions of the liver and spleen, and from pain in the upper and lower extremities along the course of the nerves. The patient's skin was a copper-red, jaundiced color; the conjunctivae were acutely hyperemic and blurred; the pupil reflex was sluggish, the tendon reflexes heightened. There was general paresthesia. The liver was enlarged, projecting from under the edge of the ribs; the spleen could be palpated and was soft and painful. The heart limits were normal, the tones somewhat muffled. The lungs were normal. Respiration was 24; pulse, 64, normal; blood pressure, systolic 120, diastolic 100. The temperature was slightly raised (37.2° to 37.8°C.) on the first and second days of illness.

Laboratory Findings.—On September 24 examination of the urine showed: color, brownish red; reaction, slightly acid; specific gravity, 1.020; albumin, 4 per cent.; Gmelin's reaction, negative; hyaline casts, granular casts in each field; three to five

fattily degenerated epithelial cells and two to three erythrocytes in each field. On September 27, the urine was slightly darkened; specific gravity, 1.017; reaction, acid; albumin, 0.33 per cent.; occasional granular casts, leukocytes, and kidney epithelial cells present in the sediment.

Examination of the blood on September 24 showed: erythrocytes, 2,570,000; hemoglobin, 52 per cent.; color index, 1.0; leukocytes, 13,400 (neutrophils, 71 per cent.; eosinophils, 3 per cent.; lymphocytes, 13 per cent.; mononuclears, 13 per cent.); proportion of leukocytes to erythrocytes, 1 to 196. Examination of the red cells showed basophilic granulation and polychromatophilia.

September 28 the findings were: erythrocytes, 2,890,000; hemoglobin, 39 per cent.; color index, 0.68; leukocytes, 9,800 (neutrophils, 59 per cent.; basophils, 1 per cent.; eosinophils, 5 per cent.; lymphocytes, 17 per cent.; mononuclears, 18 per cent.).

On October 10 the erythrocyte count was 4,610,000; the hemoglobin figure, 70 per cent. Vomiting had ceased, jaundice was diminished and slowly disappearing.

On October 27, the jaundiced color of the sclera still remained. A rapid disappearance of the liver phenomena and a slow regeneration of the blood had been occurring. Pain along the course of the nerves first increased, but by this time had disappeared.

These three cases may be considered the most acute; the remaining nine patients showed similar but less acute symptoms. In all cases the typical symptoms—shivering, nausea, vomiting, weakness, headache, and the passage of bloody urine—came on suddenly. The interval between the end of the work and the beginning of the illness varied according to the time the patient had been working, the distance between him and the etching tank, etc. But all fell ill on the day of poisoning and all within one to ten hours after leaving the works. Some of the victims fell asleep and slept quietly all through the night after the first fit of shivering, vomiting, and pain in the abdomen had passed. On the following day some of the men even tried to go out, but weakness and headache compelled them to go back to bed. Three of the poisoned men went to work two days later, but they were prohibited by us from working because they showed the typical phenomena of poisoning—hemoglobinuria, slight jaundice, weakness, etc.

Hemoglobinuria, which is caused by an extensive destruction of the red blood corpuscles, must be recognized as the most striking symptom observed in all the cases. The preliminary symptoms are, in a day or two, followed by jaundice with an enormous secretion of bile. Vomiting was observed in all the cases, was incessant in the most acute ones, and was always followed by the excretion of great quantities of thick, tar-like bile. One of the patients, who received no solid food and almost no liquids, secreted during the first days seven to eight glasses of almost pure bile.

These symptoms help us to establish the essential cause of the poisoning. Arsine belongs to the blood poisons (globulicides, according to Naegeli), particularly to the hematolytic poisons. When inhaled by the lungs, arsine passes through them without any hindrance, as through an "open gate," into the blood and produces its pernicious effect. We know that it does not remain in the lungs or damage them seriously in passing, from the fact that all the poisoned men showed either no phenomena at all in the lungs, or merely râles for a few days—a finding which did not correspond to the seriousness of the case. Post-

mortem examinations disclosed no pathologic changes in the lungs.

These clinical and pathologico-anatomic data indicate the insignificant amount of damage to the alveolar epithelium occasioned by arsine poisoning. The alveoli are penetrated as readily by arsine as they are by carbon monoxide and by atmospheric air. This immunity of the respiratory organs is an important element in distinguishing between poisoning by arseniuretted hydrogen and by nitric oxide, since the latter is always accompanied by an acute irritation of the entire respiratory tract.

Arseniuretted hydrogen in the blood acts as a powerful solvent of red blood corpuscles. A small quantity of this poison is sufficient to produce destruction of the erythrocytes, and a separating out of hemoglobin from the cell fragments. The process of cell destruction has not yet been sufficiently investigated. Albrecht and Hedinger (quoted by Naegeli (5)) suppose that the action of hematolytic poisons either damages the red corpuscles or dissolves their outer layer which contains lecithin. We must, however, suppose that the hematolytic action of arseniuretted hydrogen is not terminated when it passes out of the blood. Some of the erythrocytes are destroyed immediately; others are injured and their lives shortened—plasmotropic effect, according to Weyl (6)—the cells becoming more fragile. This explains why in nearly all the cases the amount of hemoglobin continued to decrease for a few days after the poisoning, the lowest figures being reached in three to four days' time and not immediately after the poisoning. In one of the cases (Y) the hemoglobin figure was 37 per cent. on September 24 and 25 per cent. four days later. We may possibly have here a decrease of hemoglobin already liberated from the corpuscles and not of intracellular hemoglobin, since during the first few days hemoglobinemia existed. Such an explanation is justified by two cases described by Joachim (see Naegeli (5, p. 549)), when the color index equaled 1.5 and grew normal as soon as hemoglobinuria disappeared. In some of the cases, though not always, simultaneously with the decrease in hemoglobin a decrease in the number of erythrocytes was observed during the days following the poisoning; in other cases an increase in the number of erythrocytes, but with a lowered color index, took place, with a subsequent decrease of hemoglobin. We may assume that we have here simultaneous phenomena of destruction and regeneration of erythrocytes.

Alterations in the blood in arsine poisoning are considerable, as may be seen from the cases reported. First of all, an enormous number of erythrocytes immediately disappears. In the cases described above, the lowest number of erythrocytes was 1,500,000, a decrease of 70 per cent. In the remaining cases the red cell count did not fall below 2,500,000. Out of thirty cases described by Dudley (4, p. 220), the number was below 2,000,000 in only two cases. In two cases of arsine poisoning described by Joachim (7) and terminating in recovery, the number of erythrocytes fell to 710,000 and 810,000, the hemoglobin to 16 and 18 per cent., respectively. In Case 1, the number was 2,130,000 on the first day and it had decreased to

860,000 on the ninth. It is clear that such a destruction of the erythrocytes cannot take place without some reaction on the part of the marrow, which is evidently not damaged immediately by the arsine. Signs of regeneration appear in one form or another in all the cases, manifesting themselves in basophilic granulation and polychromatophilia.

Considerable modification appears in the leukocytic system. It can hardly be supposed that the hematolytic toxins act directly on the leukocytic system as irritants. Secondary phenomena develop. The marrow is irritated by the products of decomposition of erythrocytes and hemoglobin which may be circulating in the blood—material which Naegeli calls the blood-generating irritant of the marrow. This is why a number of anemias caused by exogenous and endogenous poisons are accompanied by leukocytosis. In one case of acute poisoning by copper sulphate, Pollak observed leukocytes to the number of 62,000. In another case, one of burns of the body accompanied by a destruction of erythrocytes, this author observed an increase of leukocytes to 50,000 (see Naegeli (5, p. 549)).

In all our cases we observed the phenomena of leukocytosis, the number of white cells reaching 30,000 and 20,000 in the most serious cases. No actual parallelism between the acuteness of the poisoning and the degree of leukocytosis may be stated with any certainty. Two of the cases, with 3,190,000 and 2,790,000 erythrocytes respectively, showed comparatively high leukocytic counts—22,800 and 18,400. The increase in the number of leukocytes is due to an increase in the neutrophils. Post-toxic lymphocytosis sometimes appears when the number of leukocytes becomes normal.

Except for a leukocytosis, it is difficult to note in arsine poisoning any consistent phenomena in the appearance of the various cells of the leukocytic series. A rôle is evidently played here by constitutional peculiarities of the lymphogenous systems of different individuals, leading to different reactions to the same irritant. In two of the cases, (D) and (A), we had respectively 12,000 (51 per cent.) and 6,500 (39 per cent.) lymphocytes to 22,300 and 18,400 leukocytes. In the case of (P), there were only 1,200 (5 per cent.) lymphocytes to 20,000 leukocytes.

In spite of the vagueness of many of the changes in the hematopoietic system, the following basic elements in poisoning with arseniuretted hydrogen may be stated with certainty:

1. Destruction of and damage to red corpuscles;
2. Liberation of hemoglobin from the disintegrating erythrocytes (hemoglobinemia);
3. Myelogenous reaction, consisting in leukocytosis and the appearance of regenerating forms of erythrocytes.

All the other pathologic phenomena are connected directly or indirectly with the first and second of these effects upon the blood and may be explained by them. Hemoglobinemia causes two acute symptoms of poisoning—jaundice and hemoglobinuria. A considerable part of the hemoglobin is excreted by the kidneys. Another part is converted into bilirubin which is principally secreted by the liver as

bile and partly absorbed by the current of the blood, causing jaundice.

The bilirubin formed from the hemoglobin makes its way into the liver, which, being the organ of excretion, suffers from overstrain during this process. In the cases of poisoning which we have described the liver revealed no very marked phenomena of poisoning. It was comparatively little distended (one finger's breadth below the edge of the rib) and that only during the first days. Pain in the liver may be explained by overstrain, since it is the organ of excretion, or may be due to a considerable degree to a distention of the gallbladder by an enormous quantity of bile.

An histologic investigation made by Freifeld (8) furnishes us with a partial explanation as to where the hemoglobin and its derivatives go. Examination of the liver disclosed a marked clogging in the central portions of the bile capillaries; the bile ducts, however, were not distended. The blood vessels were unchanged and there were no phenomena of necrosis. Processes of secretion were most active, but their mechanism damaged. The bile, although not meeting with any impediments in the ducts, was clogged in the capillaries. This may be explained by the thick consistency of the bile as shown on postmortem examination in the distended and over-filled gallbladder, and in the matter vomited before death. The viscosity is caused, on the one hand, by the enormous production of bile and, on the other, by the loss of water.

The distribution of iron (hemoglobin) in the organs is still more interesting. The Kupffer cells of the liver proved to be filled with iron (hemosiderosis) and greatly swollen, as was also to some degree the reticuloendothelial system of the spleen. No iron whatever was found in the liver cells. It was the cells of the reticuloendothelial system, exclusively, that were found to be impregnated with iron; these cells being the thoroughfare of the disintegrating blood pigment on the way to its transformation.

The next station after that (the cells of the liver) proved to be laden with bile pigment. No iron was to be found between these two stations. The iron product of the blood pigment does not leave the Kupffer cells as iron. This leads us to suppose that it is in the Kupffer cells that the splitting off of the iron takes place together with the conversion of the blood pigment into bile pigment. The bile pigment is later caught by the liver cells and ejected. The fact that the liver cells act as an excreting apparatus is indirectly confirmed by all the phenomena exhibited by the liver, such as pain and enlargement which ceases with the excretion of the hemoglobin which has been converted into bilirubin.

The remaining pathologic phenomena occurring in arseniuretted hydrogen poisoning may be explained chiefly by alterations in the blood (decomposition of erythrocytes). The hemoglobin, when set free and circulating in the blood, acts as a foreign and poisonous substance (5, p. 549). It overloads all the parenchymatous organs, especially the kidneys. Moreover, arseniuretted hydrogen, in spite of the changes it undergoes in the body—changes not yet clearly understood—may possibly act as a cell poison. It may be noted, however,

that no pathologic changes indubitably connected with a toxic effect of this sort have been established. No one organ furnishes an example of any exceptional injury. The phenomena observed in our cases were of a general character and may be explained, on the one hand, by a rapid destruction of an enormous quantity of erythrocytes with the resulting effect upon the respiratory function; and, on the other hand, by the overcharging of the entire system with the products of red cell disintegration. These products of cell disintegration in arsine poisoning have not as yet been sufficiently studied. They consist of hemoglobin and its various derivatives which are split off from the decomposing erythrocytes; and of the albuminous compounds of arsenic, which, although they have possibly lost their poisonous qualities, nevertheless act as foreign matter.

All the organs of the body are charged with these fragments of shipwreck—and it now becomes the task of the body to discharge this material, some of it foreign and some of it become foreign, although originally its own. The course of the disease is a convulsive struggle of the system to free itself of this ballast. First the hemoglobin starts off in all directions; we can follow its motion to a certain extent. The kidneys and liver are principally affected, since these are the organs of excretion. Of the liver we have already spoken; the kidneys are affected to a still greater degree. They are the only outlets for the poisons (hemoglobin and the solutions of arsenical albuminous compounds), and it is easy to see that they suffer first and most of all the organs. Hemoglobinuria is one of the first symptoms of poisoning. In the renal tubules, both the straight and the winding ones, the hemoglobin precipitates out of solution, forming casts that block the ducts. Besides this, the hemoglobin and possibly the arsenical compounds injure the actual epithelium, causing the formation of casts consisting of erythrocytes, granular débris, and albumin. In acute cases this leads to a sudden breaking up of the functions of the kidneys—to anuria and oliguria of a mechanical rather than of a toxic character. The damage to the actual parenchyma of the kidneys is not very considerable. This statement is justified by the striking rapidity with which the kidneys recover. In only two of our cases did we have to deal with phenomena of a more or less protracted nephritis. In one of these, the nephritis lasted some weeks. Gross anatomic as well as pathologic-histologic examination of the body showed that the injury to the kidneys was greater than that to the other organs. It is interesting to note that the glomerular apparatus showed no modifications whatever. The straight and winding tubules suffered most. In places they were clogged with microscopic hemoglobinous casts.

The phenomena in organs other than the kidneys, especially in the nervous system (sleepiness, headache, insomnia), must be ascribed to the toxic effect of hemoglobin and of arsenic, and perhaps to a rapidly developing shortage of oxygen. In considering the nervous system, we must mention an unevenness of the pupils, observed in three of the cases. In two of them it disappeared rapidly.

The temperature level in our cases was scarcely interrupted. The inconsiderable rises observed could in most of the cases be explained by secondary kidney complications. Thus, in the case of Y, we observed a rise of 0.3° to 0.4°C. for three to four days.

The respiratory center also remained comparatively uninfluenced. The rate of respiration in the most acute case was 24 per minute; in Y's case, 20; and in the rest, still less. This fact is the more interesting since we have to deal with an enormous decrease of the oxygen absorbing capacity of the blood. We have observed a similarly slight reaction of the respiratory organs in a group of workmen poisoned by carbon monoxide. The shortness of breath, observed as a consequence of the blood's being permeated with carbon dioxide, is caused by the irritating effect of carbon dioxide on the respiratory center, and not by a lack of oxygen.

In all of our cases the heart suffered comparatively little and soon recovered. Dull tones, a somewhat quickened pulse, functional murmurs, and in some cases a slight enlargement were the only symptoms of injury.

Conclusion

These various facts elucidate the rôle of arseniuretted hydrogen. It is strictly an hematolytic poison, and by its hematolytic action almost all the symptoms of poisoning may be explained. At the same time its action is rapidly terminated. It splits up and evidently forms non-poisonous or slightly poisonous combinations with albuminous substances. The poisonous effect of the arsenic retained by the system is revealed later, when albuminous combinations of arsenic are decomposed, leading to toxic polyneurites. Polyneuritis in some of our cases developed in one to two weeks, in one case even four weeks after poisoning, and manifested itself as a pain in the nerve trunks, especially in the extremities and between the ribs, as well as in a diminished sensitiveness. There was no motor derangement.

The tenderness of the nerves observed at the beginning of the poisoning must not be confounded with these phenomena of subsequent polyneurites. The early tenderness is a phenomenon of acute toxic irritation, quickly passing away—in five or six days. After a certain interval, a polyneuritis develops which disappears in a week or ten days. The patient then slowly returns to his previous state of health.

In the cases reported we have been concerned with the question of the usual alterations in the body, and especially the blood, that develop among workmen under the influence of ordinary slight poisoning by arseniuretted hydrogen. Does a form of chronic poisoning by arseniuretted hydrogen exist? This is the question that is confronting the Institute. Cases of acute poisoning are serious, but rare. Chronic poisoning, if it exists at all, does not manifest any acute specific phenomena, but, being repeated day after day, creates a chronic pathology, perverting the physiologic functions of the blood-generating system.

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BEHAVIOR STUDIES IN INDUSTRY*

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DURING recent years, considerable interest has been manifested in the intensive study of human behavior. This interest has been focused in the main on special problems such as delinquency, both juvenile and adult, and the aberrant behavior of children and adults as observed in mental defect and disease. Although the majority of these activities have taken place in courtroom, schoolhouse, and social service agencies, there has gradually arisen a definite but growing consciousness of the need for an adequate mental hygiene. The study of the abnormal for the sake of the normal has been the stimulating aim of many. In others, the conviction has slowly grown that behavior studies in general not only will point out and explain the abnormal and the inadequate but will finally gain insight into the manifold forces that go to make up the group mind and will result in a deeper understanding of the environmental influences that surround the normal individual as well as of the influences striving within him.

It is indeed apparent that in order to gain a full and well-rounded understanding of human nature the adult in his industrial environment must be an object of study. Today our civilization is for the most part industrial; and the importance of industrial influences on human behavior must always be borne in mind. Today the family and the community are determined largely by the character and the location of the industries on which they are dependent. The economic, the social, and the political status of our population has been colored by the rapid and phenomenal development of modern industry. The average man of today is so largely dependent on his job for his well-being and happiness that his relation to his job is of great importance. What this relation is; what preparation is commonly made for it; how effectual this relationship is; in what ways the health and happiness of our workers are compromised, if at all; how our workers live and meet the obligations of their work; whether man's fundamental strivings and yearnings are satisfied by his work; what the industrial influences are that make for a better social state or tend to disintegrate our social fabric; and how our workers are adjusting themselves to these new but powerful influences—these problems are numbered among the many to be solved by those who are interested in the study of the worker. There is a definite and positive relationship between industrial life and public health which makes studies of behavior in industry impera-

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tive. Studies of human behavior necessitate the full evaluation of physical as well as of mental and social factors; and in this evaluation industrial hygiene and medicine are now ready to give great aid and to assist in the realization of an effective hygiene of the worker, the prime purposes of which are the maintenance of good health and the fostering of happy and effectual states of mind.

In an investigation of working conditions conducted some time ago in a large mercantile house of Boston which was discussed in part in an earlier paper,[1] an attempt was made to determine what methods of study are apt to prove most profitable and practicable in the industrial field, and to develop original methods of approach wherever possible. Psychologic tests were given, rating scales were devised and installed, clinical studies were made of a number of girls who were referred for adjustment of one kind or another, routine physical examinations were made of a large number of the small group studied in order to determine the practicability of rounding out the physical examinations with questions and technic of a psychiatric nature, and, finally, the investigation was closed with a questionnaire which was not to be signed with the name of the employee filling it out. These studies and experiments were carried out both in the health and in the employment departments, an opportunity which made it possible to view the problems of personnel adjustment from different angles.

The least difficulty was experienced in giving the Otis General Intelligence Examination. Here the only important obstacle was the inability to arrange for all to take the examination at one time. The girls seemed to enter into the spirit of the experiment after a few minutes, although there was some discontent while getting ready. In the installation of the rating scale there was considerable inertia and skepticism to overcome. This took a good deal of time and effort, but was to be expected. As has been previously stated, a rating scale demands careful supervision and constant attention to all the details if any weight is to be given the results. Its value as a personnel instrument, however, cannot be controverted when one considers what an improvement it is over older methods of observation and control.

The value of the clinical studies made in the health department, to which cases were referred because of some intrinsic medical problem or some difficulty of adjustment to job, cannot be overestimated. They indicate quite clearly the necessity of evaluating the physical, mental, and social factors in all cases if one wishes to obtain a true insight into the mechanisms operating toward satisfying and permanent adjustments. For illustrations of this principle one is referred to the cases cited in the paper previously published.[1]

In the attempt to include psychiatric technic in the routine physical examinations, variable results were obtained. Some of the workers were frank and accessible; others were taciturn and uncommunicative. With some it was difficult to be sure that the replies were truthfully given; a few seemed resentful. In the sphere of

[1] Elkind, H. B.: Mental Hygiene in Industry. THIS JOUR., 1924–1925, *6*, 113.

sex, considerable resistance was offered in a number of cases; therefore questions about this activity were later not included. In properly evaluating these unusual results, comparison with other studies made by the same psychiatrist is of great assistance. In his other studies the author has had much more fruitful and pleasant results, and has found resistance to the innovation much less. It must be admitted, however, that a great deal depends on the nurse who introduces the patient to the psychiatrist. If she is tactful and states the purpose of the examination fully and frankly, explaining that the psychiatrist may ask questions which one on first thought may not wish to answer, and if she urges the necessity of frankness and of complete co-operation, the results are remarkably good. This method eliminates the prude at once, and saves the psychiatrist much effort and time. In the case of minors, however, it was found best to ask as few questions about sex matters as possible. Where some definite disease process, either mental or physical, was found, or was suggested by the first examination, permission for a complete and thorough examination was obtained from the parents or the guardian. This method of approach has obviated all possible objections that might arise later.

As has been mentioned, the last experiment attempted in this store was that of the anonymous questionnaire. This was devised with a view to developing some method of approach not previously attempted which did not require the giving of a physical examination and which would secure reliable information with the least amount of disturbance to the working force or to the management and in a minimum period of time. Individual interviews are certainly time-consuming, and from this point of view alone are objectionable. Where one has to study a very large group of workers, the attempt would be very impracticable. The question of reliability is another difficulty to overcome. It seemed quite apparent that while a number of workers might be openminded to the interviewer, the majority would answer the questions of an anonymous questionnaire with at least equal, if not greater, truthfulness. The advantages of a questionnaire are, of course, many. The questions are standardized and are carefully drawn up so that ambiguous replies should be the exception rather than the rule. One can pass out a questionnaire to as large a group as is available, and the time consumed and the inconvenience caused are practically negligible. Then again the replies can be studied, carefully tabulated, and finally examined by statistical methods. One is certain, especially, that the questions asked each employee are identical—a fact which rarely is the case where individual interviews are given. This is of extreme importance if statistical significance is to be given to the conclusions derived.

By the time the specially devised questionnaire was given to the employees to fill out, the group of juniors discussed in the paper already referred to had decreased in number from about 150 to 125. The majority (111) were females, ranging in age from 15 to 24. The questionnaires were passed out to about eighty girls and

sixty-five (about 80 per cent.) were filled out and returned, giving data for 58 per cent. of the entire female group. The results of this questionnaire, together with a brief discussion, follow.

Questionnaire

Read questions carefully. Take your time. Do not sign your name. We want the information and not who gave it. When you are through enclose this in envelope and seal. Write legibly.

1. Age....years. Sex.....

Age:	15–	16–	17–	18–	19–	20–	21–	22–	23–	24 and over	*Total*
	0	6	14	13	11	6	7	2	2	4	65

Sex: All females.

2(a) How many minutes did it take you to get to the store from your house this morning? Answer.....minutes.

Minutes:	0–9	10–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	90–99	*No Answer*
	1	4	15	17	13	3	3	2	2	0	5

(b) How many minutes did it take you to get to the store from your house yesterday morning? Answer.....minutes.

Minutes:	0–9	10–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	90–99	*No Answer*
	1	5	11	19	12	4	4	0	1	1	7

3. How long did it take you to eat your breakfast this morning? Answer..... minutes.

Minutes:	0–5	6–10	11–15	16–20	21–25	26–30	*No Answer*
	3	12	23	20	1	2	4

4. Did you eat breakfast this morning (Underline which)

At Home	Boarding House	Lunchroom (near home)	Lunchroom (down town)	*No Answer*
58	2	1	1	3

5. What did you eat for breakfast this morning? (Underline the things you ate)

Meat	Fish	Milk	Butter	Bread	Coffee	Tea	Cocoa	Cake	Pastry	Eggs
5	2	7	34	56	42	13	4	7	1	35

Fruit	Biscuits	Griddle Cakes	Cereal	*No Answer*
24	8	2	27	2

6. Recreation. How many evenings did you spend away from home last week. Answer.....evenings.

Number of Evenings:	0	1	2	3	4	5	6	7	*No Answer*
	9	6	11	20	8	5	3	2	1

7. Which of the following amusements do you enjoy the most? (Underline which)

Dancing	Movies	Theater	Outdoor Sports	Night Parties	Bowling	Billiards
28	31	23	31	12	5	0

Mah Jong	Cards	Concerts	Lectures	*No Answer*
2	5	11	3	0

8. Reading. Do you read? (Underline which)

Not at All	Occasionally	A Great Deal	*No Answer*
0	41	21	3

9. What kind of books do you enjoy reading the most? (Underline which)

Biographies	Autobiographies	Travels	Detective Stories	Science	History
4	3	6	12	14	16

Love Stories	*No Answer*
44	8

10. At what hour did you retire last evening? Answer.....o'clock.
At what hour did you arise this morning? Answer.....o'clock.

(In determining the number of hours of sleep the latter was subtracted from the former)

Hours of Sleep:	5–6	6–7	7–8	8–9	9–10	10–11	11–12	12–13	*No Answer*
	2	6	17	22	13	2	1	0	2

11. At what hour did you retire the night before last? Answer.....o'clock.
At what hour did you arise yesterday morning? Answer.....o'clock.

(In determining the number of hours of sleep the latter was subtracted from the former)

Hours of Sleep:	5–6	6–7	7–8	8–9	9–10	10–11	11–12	12–13	*No Answer*
	4	5	13	17	16	7	2	1	0

12. Have you suffered today from any of the following, either slightly or severely? (Underline as many symptoms as will describe your condition. If you do not have any of the following symptoms so indicate)

Headache	Backache	Stomachache	Cold in Head	Indigestion	Cough	Weakness
6	1	0	5	1	0	0

Painful Menstruation	Nervousness	Constipation	No Complaint	*No Answer*
2	2	6	31	16

(One girl wrote "Toothache.")

13. Are you satisfied with your present job? (Underline)

Yes	No	*No Answer*
48	12	5

14. What work do you prefer? (State fully).....

(Fifty answered; 15 did not.)

1. Dress designer.
2. Clerical.
3. Present.
4. Anything better than stock girl. More money.
5. Store.
6. Store work. Just what I am doing.
7. Selling is what I like best.
8. Office work where I meet people.
9. I prefer office work, preferably something with a future.
10. Selling.
11. Clerical in department such as an assistant to buyer.
12. Clerical.
13. Would like to do some typewriting but am not quite prepared yet. Also shorthand.
14. Assistant buyer of dresses.
15. Stenography.
16. Typewriting.
17. Office work.
18. Housekeeping.
19. Nursing.
20. Office work.
21. Store work.
22. Millinery.
23. Store work.
24. Office.
25. Typewriting—office work.
26. Clerical.
27. Cashier or offices.
28. Cashier.
29. Anything but what I am doing—cashier, stock, stock-record.
30. Salesgirl.
31. Cashier.
32. Cashier. Office work.

33. Present position.
34. Cashier.
35. Cashier.
36. Present.
37. I prefer nursing but am unable to do it so I would like buying.
38. I prefer office work.
39. I don't know just what I'm capable of.
40. I want something where I am not transferred everywhere.
41. My present position—stenography.
42. Work that requires initiative.
43. Cashiering.
44. Bookkeeping preferably.
45. I prefer to become a stenographer.
46. I would rather be a stenographer or bookkeeper than running about town.
47. Secretarial work.
48. Singing. Office work.
49. Clerical work.
50. Artistic line.

15. Are you at present time taking up some special course of study in your spare time? (Underline)

Yes	No	*No Answer*
23	36	6

Kindly fold paper. Enclose in envelope and seal. Do not write your name.

Supplementary Analyses

Questions 8 and 9: Reading.—

	Biography	Autobiography	Travels	Detective Stories
Occasionally (41)	0	1	2	6
A Great Deal (21)	4	2	4	6

	Science	History	Love Stories	*No Answer*
(Occasionally)	13	11	26	7
(A Great Deal)	1	5	18	1

Question 12.—Of these six suffering from headache, only one gave additional symptoms. These were cold in head and backache. The remainder gave no other symptoms.

Questions 13 and 15.—Of those answering "no" to Question 13, eight are taking some special course of study in their spare time.

Discussion

Favorable experience with this questionnaire suggests the more common use of this method in industrial studies. Although the number of questionnaires studied is small, it is a representative sample of the whole group of female junior employees which is itself small.

In discussing the question of reliability, one has no statistical criteria from other sources with which to check the results. They seem to correspond, however, with the impressions of the store physician who has had more or less intimate acquaintance with this group for over five years. Many of the figures are self-explanatory, and only those deserving detailed consideration will be discussed here.

Question 2.—This was given in two parts practically alike in order to be somewhat self-checking. They were included because commutation to and from work appeared to be of some importance as a factor in the question of industrial fatigue. It may be safely assumed that it takes about the same amount of time to return home as it takes to go to work. The results of both questions indicate that the mode for this group falls in the class thirty to thirty-nine minutes. Approximately one-third find it necessary to

spend fifty or more minutes in getting to work. The majority spend less than forty minutes in doing so. In metropolitan cities the factor of commutation must be given some weight in the study of fatigue, especially since a fairly large proportion of the employees take two or more hours traveling to and from work.

Question 3.—This was included because it was felt that many department store employees do not take sufficient time to eat their breakfast, rising late and rushing to work. The figures here suggest that for this store, at least, this is not the case. Slightly more than a third take twenty minutes or more. More than a third take from eleven to fifteen minutes; the remainder, less than a third, take ten minutes or less to eat their breakfast. The results suggest, however, that some effort should be made through educational methods to have all employees devote at least fifteen minutes to eating their breakfast.

Question 5.—The replies to this question suggest rather definitely that the popular breakfast of the employees consists of bread and butter, coffee, and eggs; many take also fruit and cereal. This should be considered as being a rather substantial breakfast; and, if representative of what is eaten throughout the year, would suggest that the nutritional requirements are fairly well satisfied for the majority in so far as breakfast is concerned.

Questions 13, 14, and 15.—Only 20 per cent. of the group answering this question indicated that they were not satisfied with their present job. Whether this represents the true amount of dissatisfaction may be questioned. Of those answering "no" to this question, eight were taking up special courses in their spare time and this might account to some extent for their not feeling satisfied with their positions. In view of the character of the replies to Question 14, one may well assume that a large part of this seeming dissatisfaction is due to a feeling of unrest which is healthy and brought about by the various ambitions which many seem to possess. In fact, many apparently enjoy working in this store, but desire better or other positions. These results indicate plainly the need for considerable personnel work in order to effect more efficient and satisfying adjustments.

Summary

In a study of working conditions made a few months ago in a large mercantile establishment of Boston, a number of methods of approach were utilized: psychologic tests, a specially devised rating scale, clinical studies of cases referred for adjustment, routine physical examinations which included questions and technic of a psychiatric nature, and finally an anonymous questionnaire. The results of the questionnaire are here given in detail and show the value of the method and, in particular, its practicability in the industrial field from the point of view of administration.

One may conclude from a study of this questionnaire that this group of junior employees show up quite favorably. The department store employing them should feel considerable pride in the result. The majority spend not more than forty minutes in going to work and in returning home after closing time. With few exceptions they all live at home and eat a

fairly hearty breakfast. They could possibly give a little more time to eating. In spending their evenings away from home there is little to indicate indulgence in excesses. Practically all do some reading and show a healthy interest in the type of reading they pursue. They arrange their daily routine apparently in a sound manner and give a sufficient number of hours to sleep. They now and then have minor complaints but none in excess. They would do well to have periodic health examinations. The majority seem satisfied with their work and many show a healthy unrest and a desire to get ahead by taking up special courses of study in their spare time. There are definite indications for intensive, intelligent personnel work which would result in more effective adjustments to the economic advantage of both the firm and the employees.

VITAL CAPACITY OF THE LUNGS AND ITS PRACTICAL APPLICATION*

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DURING the latter half of the eighteenth century, much productive study was made of the composition of air, and especially of respired air. Some of the names associated with this work are Lavoisier, Priestly, Ruthford, and Scheele. Hutchinson (1) considered this period the second great epoch in the history of the study of respiration.

Apparently the first attempt to investigate the volume of air taken into the lungs at a single breath was made by Borelli in 1679. Within the next 100 years, Jurin, Keill, Thompson, Davy, Goodwin, Kite, and others made observations on vital capacity with the result that a wide diversity of opinion existed as to the normal vital capacity of the lungs. This is easy to understand when it is considered that no standard instrument had been devised for measuring accurately the vital capacity and that the observations of most workers were limited to a very small number of persons. In some instances only a single case was reported, and the size and the condition of the body were apparently given little consideration.

Seemingly it was left to Hutchinson (1) to place on record the first painstaking and scientific investigation of the vital capacity of man. His paper, published in 1846, contained illustrations and descriptions of the spirometer which he had invented and, in addition, presented the results of his observations of approximately 3,000 people, some of whom were in good health while others were suffering from diseases of the lungs. Hutchinson's work also paved the way to the accurate measurement of the tidal air, the supplemental air, and the complemental air, to the calculation of the residual air, and, later, to the collection of respired air for basal metabolism determinations, etc. This may be considered the beginning of the third great epoch in the history of the study of respiration. That Hutchinson was a true scientist cannot be doubted after one reads the

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following concluding paragraphs of his paper.

> The matter of this communication is founded upon a vast number of facts—immutable truths, which are infinitely beyond my comprehension. The deductions, however, which I have ventured to draw therefrom, I wish to advance with modesty; because time, with its mutations, may so unfold science as to crush these deductions, and demonstrate them as unsound.
>
> Nevertheless the facts themselves can never alter, nor deviate in their bearing upon respiration—one of the most important functions in the animal economy.

Hutchinson stated that the vital capacity of man may be considered as a constant quantity, but that this quantity is directly disturbed or modified by four circumstances—namely, height, weight, age, and disease.

During the next fifty years, Simon (2), Fabius (3), Wintrich (4), Arnold (5), Müller (6), Waldenburg (7), Cornet (8), and others made extensive studies in vital capacity.

It was during the world war that the vital lung capacity test found one of its most useful periods. For various branches of the service, men were needed in great numbers and time did not always permit a careful and complete physical examination; any test, therefore, which would quickly eliminate those afflicted with serious conditions of the heart and the lungs was of great value. Dreyer (9), working with the British forces, pointed out that the vital capacity test was being used as a routine in the examinations of candidates for the British Flying Service and that it served as a decisive factor in election to or rejection from that service. It was this experience which stimulated Dreyer to investigate further the subject of vital capacity and to report his valuable findings on the subject. Schneider, working with the American forces at the same time, made considerable use of the vital lung capacity test.

Already the vital capacity fire was burning in America, for Peabody and Wentworth, West, and others of Harvard were investigating this subject and publishing the results of their splendid work. In the fall of 1920 Professor Dreyer spent considerable time lecturing on this subject in various medical centers of America. So stimulating and convincing were his lectures that numerous investigations in various parts of this country followed in his wake. These investigations have been concerned not only with the vital capacity of normal individuals but also with the effects of certain diseases on vital capacity, with the result that many findings of the earlier writers have been confirmed and a good many new facts have been and will continue to be added to our store.

Normal Vital Capacity Standards

It is obvious that before any test can render its greatest service reasonably accurate normal standards must be developed for comparative purposes. Since Hutchinson began his work on vital lung capacity several normal standards have been presented, such as the standing height standard, the sitting height standard, the chest circumference standard, the

surface area standard, and the body weight standard. We shall consider briefly the development of the more important of these normal standards and their relative value in the light of present-day investigations.

Standing Height Standard.—West (10) has concluded that "For quick clinical work, where it is not practicable to obtain the net weight of the patient nor to make extensive measurements, a simple comparison between the vital capacity and the height will give a value which will closely approximate the normal for that individual, though individual variations by that method are greater than when the surface area is used." West found that for every centimeter in height a man gains 25 c.c. of air, while a woman gains only 20 c.c. Bowen and Platt (11) and others have found these ratios quite satisfactory. Our tables were prepared from the ratios given by West. Boynton (12), Shepard (13), Maeder (14), Cady (15), and Myers (16) have applied these tables to good-sized groups of normal men and women and have obtained quite satisfactory results. One advantage in using the standing height standard is that it is not affected by the obesity of the subject.

Surface Area Standard.—In 1917 Peabody and Wentworth (17) conceived the idea that a close correlation exists between the vital capacity of the lungs and the surface area of the body. They made a few observations which proved very satisfactory, whereupon they suggested that a study be made on a larger number of individuals. West (10) took up this problem and in 1920 published his results. He found that for each square meter of body surface men have a vital capacity of 2.5 liters, while women have a vital capacity of only 2 liters. At the present time this method of West's is becoming quite generally adopted because it is easy to use and apparently is as accurate as any method in use.

Body Weight Standard.—In 1919 Dreyer (9) presented a formula for obtaining the normal vital capacity. This formula is based on body weight, but body surface area is given due consideration.

After the examination of more than 12,000 normal persons at the University of Minnesota, we have arrived at the conclusion that the surface area and body weight methods of West and Dreyer are most satisfactory in obtaining the theoretical normal vital capacity of a given person. It matters little which of these methods is used. In order to save time for the physician we have constructed tables (18) (19) from which one may quickly obtain the percentage of the theoretical normal vital capacity which any given person presents.

Factors Other than Disease Which Influence the Normal Vital Capacity of the Lungs

Physical Training.—In using the normal vital capacity tables one finds a few persons who present vital lung capacities of as much as, or more than, 125 per cent. of the normal. Usually these are people who have done hard labor, such as farm work, or who have been active in athletics, or have trained the voice for singing, or have played a wind instrument

throughout a long period of time, or have indulged in some other activities which have led to an over-development of the lung capacity. Such individuals with over-developed lung capacities represent a more important group than one is apt to believe at first thought. Members of the group, after developing diseases of the heart or the lungs which decrease their vital capacities, may still have capacities of more than 100 per cent. of the theoretical normal. The mere fact, therefore, that a given individual presents a vital capacity of 100 per cent. or more of the theoretical normal does not prove that that individual is free from heart or lung disease which may indeed have definitely decreased his vital capacity.

Occupation.—Ever since vital capacity has been studied it has been said to be materially influenced by occupation. Hutchinson grouped his cases according to their occupation, as did numerous succeeding investigators. Dreyer (20) believes occupation to be so important that he groups all people into three classes, which he designates as A, B, and C.

Foster and Hsieh (21) found that "Occupations seem to influence the vital capacity to some extent, although not in proportion to the degree of physical activity." It must be borne in mind that, while occupation plays a considerable rôle in the vital capacity of a given case, there is much individual variation in vital capacity and that not all persons in a given occupation have uniformly low or high vital capacities.

Sex.—Fabius (3) and Wintrich (4) pointed out that a definite difference exists between the vital capacities of men and women. Wintrich found that the sex variation is very slight during the early years but becomes more noticeable after the fifteenth year of life. Stewart (22) has shown graphically that from the age of 4 years the vital capacity of boys is constantly slightly greater than that of girls until about the age of 14 years, when the sex difference becomes more marked. Wilson and Edwards (23) found that the vital capacity of boys is 6 per cent. greater than that of girls.

In senile individuals Wintrich (4) found that in general the sex difference continues up to the greatest ages. This finding was recently confirmed by Myers and Cady. The differences due to sex between young adult life and senility are such as to require separate normal standard tables for the two sexes.

Age.—Hutchinson (1) believed that the maximum vital capacity of an individual is reached about the thirtieth year of life. Stewart's (22) recent studies led him to believe that the maximum vital capacity of the lungs is reached in boys about the twentieth year and in girls slightly earlier than that time.

Wintrich (4) pointed out that the ratio between the standing height and the vital capacity varies with the different ages of childhood and differs markedly from that in adult life. Recently, Stewart (22), Wilson and Edwards (23), and Emerson and Green (24) have made extensive studies on the vital lung capacities of children and have shown that standards used for adults are not applicable to children.

Peabody and Wentworth (17), West (10), Pratt (25), McKean (26), Bowen and Platt (11), and Lemon and Moersch (27) have called attention to a decrease of vital capacity in old age. Myers and Cady (28) observed that in those individuals who continue to lead active lives the vital capacity does not decrease so much as in those who retire from work at the age of 50 to 60 years. They believe that those who do not retire until unfortunate physical conditions compel them to do so have a higher percentage of physical fitness and lead much happier lives than their retired friends.

Obesity.—Hutchinson (1) taught that an excess weight of 7 per cent. or more interferes with the vital capacity of the lungs. Recent investigations lead one to believe that only extreme obesity is capable of actually reducing the vital capacity, and then because of the deposit of fat in the abdomen and thorax which may limit somewhat the diaphragmatic excursion and thus decrease the capacity of the thorax. It has been stated (11) that in dealing with obese individuals one may use the height standard, or, if the surface area standard is used, 20 per cent. should be added to the observed vital capacity.

Posture.—Christie and Beams (29) found that when the subjects were in a reclining position the vital capacity averaged 5.5 per cent. lower than when they were in the sitting posture. Rabinowitch (30) recently investigated this subject and found that if the vital capacity observed in the recumbent position is multiplied by the constant 1.075 the product may be accepted as the vital capacity which the same individual would present in the standing position. This is an important subject, inasmuch as one is frequently compelled to examine acutely ill patients in the recumbent position and should know, therefore, approximately how much of the reduction in vital capacity is due to posture.

Race and Nationality.—Wilson and Edwards (31) examined a group of thirty-eight average normal colored children ranging from 8 to 14 years of age. They found the vital capacity strikingly below that of any other group studied and stated that "these findings suggest a possible racial factor." Foster and Hsieh (21) made observations on 500 Chinese in several occupations, including a number of young women. They found that "the Chinese show much lower vital capacity ratios than Westerners." The work which Dr. Concepcion (32) has under progress at Manila promises to throw much light on this subject. In small groups of Chinese and Philippinos, I have found the vital capacities lower than among the white race.

Dreyer (20) says, "If a person is found to have as much as 10 per cent less vital capacity than is normal for his class, it is probable that he is suffering from some health depressing condition and if he is as much as 15 per cent below the normal limit, it is *practically certain* that he is abnormal in this respect."

After a careful study of a group of children, Wilson and Edwards (31) state that "A reduction of vital capacity measurement of 15 per cent, or more, from the average normal

standard should single the child out for further physical and roentgenographic examination."

Disease Factors Which Influence the Vital Capacity of the Lungs

It has been proved that disease of certain organs besides the lungs, and particularly of the heart, is capable of reducing a person's vital lung capacity. Abnormal conditions within the chest which decrease the vital capacity may be classified as (1) those of present clinical significance, and (2) those of no present clinical significance.

Diseases of Present Clinical Significance

Some of the diseases of present clinical significance which decrease vital lung capacity are: cardiac disease, bronchitis, pleurisy, pneumonia, pulmonary tuberculosis, and pneumothorax.

Cardiac Disease.—Von Basch (33), Rubow (34), Siebeck (35), Peabody and Wentworth (17), Levine and Wilson (36), Adams and Sturgis (37), Ulrich and Nathanson (38), Burton-Opitz (39), Pratt (25), Lattimore (40), Ziskin (41), and Levison (42) have made valuable contributions to our knowledge of vital capacity in heart disease. The work of these investigators has proved that vital capacity readings aid in diagnosis, in prognosis, and in guiding the treatment. It has been pointed out (Ulrich and Nathanson) that in patients complaining of cardiac symptoms the vital capacity aids in determining the presence or absence of cardiac involvement. Some striking cases are cited of patients who appeared to have marked symptoms of cardiac disease, yet their vital capacity was normal and further examination proved the absence of cardiac disease. Adams and Sturgis (37) found that in 100 patients with "effort-syndrome," complaining of dyspnea, the vital capacity was within normal limits. Levison (42) says, "The vital capacity of cardiac patients runs roughly parallel to their dyspnea and this fact may be utilized in differentiating true from false dyspnea."

On the other hand, when cardiac disease does exist the vital capacity test gives reasonably accurate information concerning the functional capacity of the heart. Peabody and Wentworth (17) and Ulrich and Nathanson (38) have shown that the reduction in vital capacity closely follows the clinical condition of the patient, varying directly with the degree of cardiac impairment. It has been stated that, as a rule, the lower the vital capacity, the poorer the prognosis. This does not hold true in every case, however, since certain patients with acute decompensation may show a very low vital capacity which increases materially as compensation is established. Other patients, on the verge of developing decompensation or other conditions which usually warrant a grave prognosis, may have a good vital capacity. This represents the actual functional capacity of the heart at the time the reading is taken, but yields no information as to the progressiveness of the lesion. Subsequent readings, however, may show a gradual decrease in spite of treat-

ment. Such findings usually warrant a bad prognosis.

Ulrich and Nathanson (38) believe that possibly the greatest aid which the vital capacity test offers in cardiac cases is in relation to treatment: "It (1) gives us a measure of the results of treatment; (2) indicates the maximum point of response to treatment; (3) establishes a cardiac tolerance for work; and (4) shows whether this cardiac tolerance for work is increasing or diminishing." They believe further that just as we frequently examine for glycosuria in cases of diabetes in order to determine whether the patient is remaining within his carbohydrate tolerance, so also we may frequently apply the vital capacity test in cardiac cases in order to determine whether the patient is remaining within his cardiac tolerance.

Pratt (25) says: "The graphic records on the charts of these and similar cases show the value of long continued observations of the vital capacity repeated over a long period of time in measuring the increase or decrease of cardiac power. They will be found to be a distinct aid in the treatment and prognosis of heart disease."

Stewart (22) found that "Children may have a normal vital capacity even in the presence of a definite insufficiency of the mitral valves, and these individuals show little abnormal tendency to dyspnea on exertion. In more serious cases the vital capacity is greatly reduced, and the tendency to respiratory embarrassment is proportional to the reduction in lung capacity. When the condition of the heart improves, the vital capacity gradually increases."

Wilson and Edwards (43), after studying the vital capacity of children with heart disease, concluded that "Vital capacity measurements show a close relation to the heart functional capacity, as gauged by the exercise tolerance. They cannot be considered quite so reliable an index of diminished capacity for physical activity in children with heart disease because of the wide variation obtained in the normals and the chance of other complicating factors."

Bronchitis.—In 156 cases of chronic bronchitis, Myers and Cady (44) found that the average vital capacity was 101.1 per cent., while in twelve acute cases it was 100 per cent. of the theoretical normal. In 162 cases with peribronchial infiltration revealed by the X-ray the average vital capacity was found to be 101 per cent. of the theoretical normal. Thus, it appears that such conditions decrease the vital capacity very little or not at all.

Peabody and Wentworth (17) found that the vital capacity may be reduced to a considerable extent during an attack of *acute bronchitis*. In children suffering from acute bronchitis, Stewart (22) observed a temporary reduction in vital capacity. He points out that the presence of considerable mucus in the tubes often provokes coughing toward the end of expiration and thus prevents complete expiration.

Pleurisy.—In acute fibrinous pleurisy the vital capacity may appear to be greatly decreased because of the severe pain which makes it im-

possible for the patient to take a deep breath. Severe acute fibrinous pleurisy, accompanying such conditions as pneumonia, pulmonary abscess or pulmonary carcinoma, often lowers the reading very greatly.

In cases of serofibrinous pleurisy, the reduction in vital capacity depends to a large extent on the amount of fluid in the pleural cavity. In purulent pleurisy Graham (45) showed that it is greatly reduced. "That this reduction does not depend merely upon the presence of the fluid exudate in the pleural cavity is shown by the fact that an appreciable increase in the vital capacity occurs only gradually after the removal of the exudate."

It has been shown recently by Sturgis, Peabody, and Hall (46) that when the chest is bound down with a swathe the vital capacity may be reduced approximately 50 per cent. and the maximum minute-volume about 50 per cent. In this work it was demonstrated that the subjective sensation of dyspnea increases as the actual minute-volume encroaches upon the maximum minute-volume. The condition produced in this work is believed to simulate closely that in pleurisy with effusion where the vital capacity is reduced. "The essential cause of the variation which may occur in the maximum minute-volume is an alteration in the vital capacity of the lungs and this may, therefore, be regarded as a practical index of the maximum minute-volume."

Pneumonia.—Peabody and Wentworth (17) reported five patients convalescing from pneumonia in whom the vital capacity varied between 71 and 114 per cent. of the normal. After a study of some twenty cases Myers (47) stated that there is probably no acute disease of the respiratory tract which so reduces the vital capacity from the beginning as does pneumonia. The vital capacity test is, therefore, a valuable aid in early diagnosis. It has been possible to take vital capacity readings on patients suffering from acute respiratory infections (other than pneumonia) and other acute diseases with symptoms closely simulating those of pneumonia and to select those developing this disease from the vital capacity test alone. In pneumonia the vital capacity is reduced tremendously and the reduction is not consistent with the extent of involvement; persons with disease limited to one lobe or even a part of one lobe may have a vital capacity as low as those with much more extensive involvement. Usually the maximum reduction is reached on or near the day of the crises. After this time, if convalescence is undisturbed, the vital capacity supplies a valuable index as to the clearing up of the lesions. In cases of unresolved pneumonia or in the development of such complications as lung abscess or empyema the vital capacity increases very slowly or not at all. This test is extremely valuable in determining when it is safe for a patient convalescing from pneumonia to become active. Patients are too often discharged from the hospital and returned to active lives before their lung capacities are consistent with such activities. It is recommended strongly that no patient undertake

an active life either in the hospital or in the home until the vital capacity has been restored to within 90 per cent. of the normal.

After a careful study of twelve patients suffering from pneumonia, Arnett and Kornblum (48) stated that their observations seemed to indicate the following uses for the spirometer in this disease.

(*a*) As an adjunct to the usual clinical pulse, temperature and respiration record, the vital capacity graph assists in visualizing the course of the disease. (*b*) After the crisis, a failure of the vital capacity to increase rapidly and steadily may point towards a complication, while the presence of such an increase argues against the latter. (*c*) Frequent accurate vital capacity readings before and after operation for empyema are apparently valuable in indicating the success or failure of the operation. (*d*) An extremely low vital capacity may assist in differentiating pneumonia from diseases which do not lead to such a marked reduction.

In children Stewart (22) found that lobar pneumonia affects the vital capacity of the lungs in a manner very similar to that above described for adults.

Pulmonary Tuberculosis.—In 1846, Hutchinson (1) reported that he had been greatly aided by the vital capacity test in the diagnosis of pulmonary tuberculosis. Following Hutchinson's work, Wintrich (4), Simon (2), Arnold (5), Fabius (3), Schneevogt (49), Müller (6), Waldenburg (7), Cornet (8), and others found the vital capacity definitely reduced by pulmonary tuberculosis. Shepard (50), after using the vital capacity test very extensively in the diagnosis of tuberculosis during the past forty years, states that he has found it of very great value. Garvin, Lundsgaard, and Van Slyke (51), in addition to other valuable findings, learned that in minimal tuberculosis the vital capacity was decreased as a result of an increased amount of residual air, while in moderately advanced cases the decrease was chiefly due to a diminished total capacity. Dreyer and Burrell (52), from a study of more than 200 cases of pulmonary tuberculosis, found the vital capacity test valuable in diagnosis, in classification, and in following the results of treatment.

Myers and Rice (53) called attention to the value of the vital capacity test in the diagnosis of pulmonary tuberculosis. A comparison of vital capacity readings with X-ray findings in 717 cases showed that when X-ray findings were negative the average vital capacity was 104.1 per cent. Two hundred and three patients with slight unilateral parenchymatous lesions had an average vital capacity of 98.7 per cent., while seventy with slight bilateral parenchymatous lesions had an average vital capacity percentage of 95.5. In ninety-four cases with unilateral disease involving not more than one lobe, and in ninety cases with bilateral disease involving not more than two lobes, the average vital capacity percentage was found to be 96.8 and 80, respectively. Twenty-two patients with unilateral disease involving more than one lobe and forty-six patients with bilateral disease involving more than two lobes had average vital capacities of 65.6 per cent. and 53.8 per cent., respectively. It was of interest and of practical value to find that of the 203 cases with slight

unilateral disease revealed by the X-ray 152 proved to be non-clinical. Their average vital capacity was found to be 103 per cent. of the normal. Only forty-two of the 203 cases had clinical tuberculosis. Their average vital capacity was 85.5 per cent. of the normal. There were nine cases with questionable activity with an average vital capacity of 89 per cent. of the normal. It was found that throughout the various groups where non-clinical tuberculosis existed the vital capacity was approximately normal, whereas in the presence of clinical tuberculosis the average capacity was very definitely reduced.

When the vital capacity readings, symptoms, and physical findings were correlated in 773 cases, it was found that when the physical signs and symptoms were negative the average vital capacity was 103 per cent. In cases of pulmonary tuberculosis with physical signs of parenchymatous involvement the average vital capacity was found to be decreased as the physical signs and symptoms increased.

Cameron (54) recently reported the results of his observations on 198 tuberculous patients. He found the correlation between the vital lung capacity and the anatomic changes in the lungs so close as to show convincingly that vital capacity readings deserve a definite place as a supplementary method of diagnosis and prognosis.

Contrary to what one might expect, those who have made studies of the vital capacity of the tuberculous state that they have seen no harm result to patients from this test.

Pneumothorax.—Pneumothorax, whether artificial or spontaneous, causes a decrease in vital capacity. In studying chronic pulmonary disease, one occasionally finds a case in which vital capacity quite suddenly drops several hundred cubic centimeters. When this occurs, spontaneous pneumothorax should be suspected. In artificial pneumothorax the vital capacity reduction apparently does not depend entirely on the amount of gas injected into the pleural cavity. For instance, an individual with a vital capacity of 3,000 c.c., after having 500 c.c. of gas injected, may blow more or less than 2,500 c.c. In a small series of cases of pneumothorax reported by Myers (47), the average vital capacity was found to be 49 per cent. of the normal. In a recent study by Myers and Bailey (55), vital capacity readings have been found of considerable value in guiding the administration of artificial pneumothorax. In some cases it has been found that the initial injection of gas, although it may be small, reduces the vital capacity a great deal. For example, one patient with a capacity of 3,200 c.c. had the capacity reduced to 2,300 c.c. after the injection of 250 c.c. of gas. Before giving the initial injection of gas, therefore, one should know the patient's capacity. If this is low, only a small amount of gas may reduce the capacity to or even below the tidal air; thus resulting in dyspnea and much distress to the patient. In our cases, it has been shown that with subsequent injections of gas the vital capacity usually is not reduced even to the extent of the amount of

gas injected. This is especially noticeable in cases in which there is extensive disease of the lung under treatment, while the opposite lung is clear.

With the worker who has had little experience in pneumothorax it sometimes happens that the needle passes into the lung because the visceral and parietal pleurae have become adherent. Slight oscillations on the manometer may lead him to believe that the needle is in the pleural cavity and a considerable amount of gas is injected. In such cases, the vital capacity reading is the same before and after the attempt to induce pneumothorax.

If vital capacity readings are taken in every case before and after each injection of gas the readings will be found valuable in determining the condition of the better lung. Thus we have been convinced that routine vital capacity readings are of practical value in pneumothorax cases.

Surgery of the Thorax.—After making a careful study of the importance of vital capacity in thoracic surgery, Graham (45) arrived at the following conclusions:

> Determinations of the vital capacity by means of a spirometer, when used in connection with the mathematical expression given in the text, will indicate approximately the maximum opening in the chest wall compatible with life, if the mediastinum is not already stabilized by adhesions and induration. If such observations are made before establishing open drainage in cases of empyema or before any thoracic operation, doubtless many lives will be saved. Both theoretical conclusions and actual observations show that in empyema the vital capacity is greatly reduced. That this reduction does not depend merely on the presence of the fluid exudate in the pleural cavity is shown by the fact that an appreciable increase in the vital capacity occurs only gradually after the removal of the exudate. This fact is of importance in being an additional argument against the establishment of an open drainage during the acute pneumonic stage of an empyema when the vital capacity is so low as to approximate the tidal air requirement. Extensive thoracoplastic operations result in apparently a permanent marked reduction in the vital capacity. They should be employed, therefore, only in the rarest instances and only after other methods have been given an intelligent trial for at least many months.

After making a study of the vital capacity of 579 surgical cases, estimating the risk and later comparing their estimate with the surgeon's estimation of the risks and later observing the postoperative course, Lemon and Moersch (56) arrived at the following conclusion:

> For routine purposes, the determination of the operative risk from the surgeon's opinion is probably, on the whole, a more satisfactory estimate than that obtained by means of the vital capacity readings. However, if the surgeon does not have the benefit of experience, or the assistance of an experienced internist in making such an estimate, especially in questionable cardiac and pulmonary conditions, vital capacity determinations may be of the greatest aid. From whatever cause, the closer the approximation of vital capacity to tidal air, the graver will the risk become. When low readings are obtained, the surgeon would do well to institute preoperative measures, and to make further observations before attempting operation.

Past Diseases

In the past there has been considerable speculation as to whether vital capacity, once reduced by certain diseases, ever returns to the normal. Recently, Shepard (57)

made an extensive study of this subject in which he used 611 male university students with histories of influenza, pneumonia, pleurisy, pulmonary tuberculosis, or heart disease. He compared the vital capacities of these students with those of 2,821 students without histories of such diseases. From his study, Shepard concluded that "Influenza and pneumonia appear to lower the vital capacity of a large group, although the change is slight. Pneumonia varies but slightly in its effect on the vital capacity, according to the age contracted. When pleurisy, pulmonary tuberculosis or organic heart disease is included among the past diseases of a group of persons, there is apparently a definite and probably a permanent lowering of the vital capacity of that group as a whole."

Application of Vital Capacity Test

The greatest value of the vital capacity test is realized when one is able to take serial readings on each case over a considerable period of time. I have found such serial readings (58) very helpful in determining whether the lung capacity increases consistently with the growth of the bodies of children suspected of having pulmonary pathology. They are also very helpful in diagnosing cases developing pulmonary pathology subsequently to the recording of the initial reading, and in following the progress of treatment of lesions in the heart or the lungs.

In this connection, the vital capacity test has an important place in industrial and preventive medicine. Industrial organizations will find that routine vital capacity determinations on all persons entering their employ will greatly aid in the detection of certain diseases which might otherwise be overlooked. Again, if such readings are kept on record, they will often be found valuable for comparison with subsequent readings taken at times when employees may be developing early disease of the heart or lungs, or when claims for compensation are made because of physical disabilities supposedly developed while in the company's employ.

Many colleges and universities are now making vital capacity readings a routine procedure in the entrance examinations of all students registered. Already clinicians have found such records valuable. Readings are being made also in many schools, and the matter of making them a part of the routine physical examination of schoolchildren was discussed favorably at a recent convention of the National Education Association.

The time is at hand when persons should know their actual vital lung capacities, just as they should know their actual heights and weights. Any person who loses several pounds in weight has reason to seek the cause; so, also, any person whose actual lung capacity decreases more than 10 or 15 per cent. has just cause for seeking investigation. Many persons would become familiar with their actual vital lung capacities if spirometers were made available as weighing scales are. The installation of accurate spirometers and tables with normal vital capacities and weights in many places where

they would be available to the public would be an excellent public health measure.

What Causes the Vital Capacity to Be Reduced

This question has been asked frequently, and a number of explanations have been offered. Von Basch (33), Siebeck (35), Ulrich and Nathanson (38), and Barron (59) call attention to the fact that early in *impaired cardiac function* there is a stasis and enlargement of the capillaries in the walls of the lung alveoli. These changes, they believe, affect the elasticity and expansibility of the alveolar walls. And, although Mackenzie and others have considered râles at the bases of the lungs as one of the earliest evidences of cardiac decompensation, these observers further point out that "a reduction in vital capacity, although an expression of the same engorgement process, occurs, however, at an earlier stage, and may therefore be taken as an earlier evidence of cardiac impairment." The work of Drinker, Peabody, and Blumgart (60) has shown that intravascular blood is capable of obstructing and encroaching upon the alveolar spaces, thus reducing their capacity.

About *pulmonary tuberculosis* it has been known for some time that with even a small lesion the expansion of the chest and the movements of the diaphragm on the same side may be considerably limited. It has been shown also that a severe lesion, even if very small, may reduce the vital capacity more than an extensive lesion of a milder nature. Garvin, Lundsgaard, and Van Slyke (51) have shown that in such cases the reduced vital capacity is due to increased residual air. These findings would suggest that a change occurs in the walls of the alveoli, which limits the use of the affected lung. In moderately and far advanced pulmonary tuberculosis, the destruction of tissue undoubtedly plays a considerable rôle in diminishing the vital capacity. In fact, Garvin, Lundsgaard, and Van Slyke found that in moderately advanced pulmonary tuberculosis the reduction in vital capacity is due largely to a diminished total capacity.

In *pneumonia* it has been found that the reduction in vital capacity is not proportionate to the extent of involvement. For instance, in a case with a small area of involvement there may be a vital capacity reduction as great as in a case with consolidation of one or more entire lobes. Perhaps the compensatory emphysema in the lobules adjacent to the affected ones and the congestion of the alveolar capillaries, together with the limited movement of the diaphragm on the side of the involvement, account for the greater part of the reduction.

In *asthma* the marked reduction in vital capacity during acute attacks is apparently due to obstruction caused by constriction of the bronchial musculature. Obstruction of the tubes by an accumulation of mucus undoubtedly is responsible for the greater part of the reduction in occasional cases of *bronchitis*.

It was suggested by Peabody and Wentworth (17) that the reduction in patients convalescing from pneu-

monia is due possibly to weakness following toxemia and may be found in patients convalescing from any acute toxic condition. My observations (47) on the vital capacity of sixty patients suffering from and convalescing from *paratyphoid fever* showed that the vital capacity is reduced below normal limits in a very small percentage of the cases, and in these cases the reduction could be explained on other bases, such as development of pleurisy or pneumonia. During convalescence, the vital capacity of the uncomplicated cases was found to be within normal limits. In this series of cases fever and rapid pulse had apparently little or no effect upon the vital capacity.

It has been stated that the vital capacity test is a good index as to physical fitness, but some of the more recent work suggests that a correlation of vital capacity with certain measurements of the body is an index of physical fitness only in relation to the heart or the lungs. Peabody and Sturgis (61), studying the effects of general weakness on the vital capacity of the lungs, found twelve patients suffering from anemia all of whom gave history of weakness, some in the extreme form. The average vital lung capacity of these patients was 88.7 per cent. of the normal; the lowest, 74 per cent. of the normal. Another patient on whom "test made on thirty-sixth day of typhoid fever and second day of normal temperature, who could not register grip testing apparatus with either hand," had a vital capacity of 86 per cent. of normal. It would appear that these figures could hardly represent the true physical fitness of the patients studied.

Peabody and Sturgis (61) made a further study of the effect of fatigue of the muscles of respiration on the vital capacity. They attempted to produce this fatigue by taking vital capacity readings every fifteen seconds for ten minutes. The individuals used in this work were suffering from cardiac disease or pernicious anemia. It is interesting to note that they showed a vital capacity at the end of the experiment as great as at the beginning.

It appears, therefore, that patients suffering from a disease outside the heart and the lungs which causes an extreme weakness have a reduction in physical fitness greater than the vital capacity test indicates.

Conclusions

While normal vital capacity standards are as satisfactory as normal weight standards, yet just as is the case with weight standards, individual variation offers one of their greatest limitations. A person who may ordinarily have a vital capacity of 125 per cent. of the theoretical normal may develop a disease which materially reduces his vital capacity, and yet the readings may be well within the generally accepted normal limits. This difficulty is overcome where the vital capacity of persons while in a normal state of health is recorded.

In the differential diagnosis of chest diseases the vital capacity test gives us very little aid. Probably the only help we receive from it is in differentiating pneumonia from certain other acute diseases.

Contrary to fairly recent teaching, it is apparently a test of physical

fitness only in so far as the heart and the lungs are directly involved.

On the other hand its uses are many and very definite. It can tell us roughly, yet quickly, whether there is involvement of much clinical significance affecting the heart or the lungs directly or indirectly. It aids in differentiating between pneumonia and certain other acute diseases of the respiratory tract. It gives valuable information regarding the severity of lesions, especially tuberculous lesions. In surgery of the thorax, including artificial pneumothorax, it has a definite place.

In dealing with acute infections outside the heart and the lungs, the vital capacity test is valuable in detecting developing complications such as pneumonia. The test is a good index also as to the effects of treatment of diseases of the heart and the lungs. If improvement occurs the vital capacity may show an increase before any change can be detected by other methods of examination. On the other hand, if the condition remains stationary or progresses, the vital capacity will remain unchanged or show an actual decrease.

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Occupational (Professional and Industrial) Injuries of the Skin. Edited by *Karl Ullmann*, *Moriz Oppenheim*, and *J. H. Rille*. Volume I. Pp. 336. Leipzig: Leopold Voss, 1922.

This book consists of two volumes. Volume II, which has not yet reached us, will deal, we understand, mainly with the injuries of the skin caused by chemical poisons used or handled by professional and industrial workers during the performance of their duties. Volume I, edited by Dr. Ullmann and Dr. Rille, is written in German and deals mainly with the injuries of the skin due to physical causes. It contains eighteen separate articles by authorities of various nationalities, most of whom have their headquarters either in Austria or in Germany. At least several of the authors are well known throughout the realm of dermatology and its kindred sciences.

Bernardino Ramazzini, who was born in Carpi in 1633 and died in Padua in 1714, is regarded as the founder of industrial hygiene, and his portrait appears as the frontispiece to this volume. A general outline of the etiology and nature of industrial skin affections is given under the name of the late A. Blaschko. J. Fischer deals with the history of industrial dermatology. Dr. Moriz Oppenheim writes on vocational stigmata of the skin, a subject for which he is already famed. These stigmata afford indications to a man's occupation and particularly to the tool or material—chemical, oil, dye, etc.—with which he habitually works. Two colored plates and numerous excellent black and white illustrations of callosities of the hand, etc., are given.

The article on the pathology and clinical features of the action of cold is written by Dr. Kyrle, who deals chiefly with *erythema pernio* (chilblain) and genuine freezing of the skin as seen, for instance, in the feet of soldiers exposed to low temperature during the recent war. He treats fairly fully with the predisposing factors, prophylaxis, and therapeutics. Several typical illustrations, including a colored plate, demonstrate the chief points discussed. Dr. Ullmann contributes a complete and lucid description of the effect of heat in producing burns and scars of the skin, dealing with statistics and with stigmata showing, for instance, the characteristic position of burns in various occupations—*e.g.*, baking, casting, metal working, etc. Numerous photographs of injuries and also of interiors of foundries illustrate his discourse; preventive measures and treatment are duly considered. Dr. Jellinek writes concerning the injuries to the skin produced by electric shock; his article is suitably illustrated and includes notes on diagnosis, preventive measures, and therapeutics. Dr. Ehrmann and Dr. Perutz give an account of experiments dealing with the rudiments of the pathologic effects of light rays; while sunburn, *hydroa vacciniforme*, and the graver condition of *xeroderma pigmentosum* are fully described by the late A.L. Fönss of the Finsen Institute. Typical illustrations accompany these descriptions and reference is made to the various conditions of sensitization to

luminous rays by such substances as hematoporphyrin and buckwheat; Hausmann's experiments on sensitization of mice to visible rays by injection of the former substance receive attention. The effects of mercury vapor and carbon arc lamps also come under discussion. Three interesting articles accompanied by excellent illustrations concern injuries of the skin produced by special radiations: Dr. Dohan and Dr. Kienböck deal with roentgen rays, Dr. Dautwitz with radium, and Dr. Friedländer with mesothorium.

A rather novel article by Dr. Silberstern describes skin injuries produced by the formation of gas bubbles (nitrogen) in the skin of compressed air workers; reference is also made to skin affections following indirectly upon injury to the spinal cord by these gas bubbles. Dr. Silberstern also describes accidents to divers in which, owing to the much greater pressure of the water on the body than on the helmeted head, blood is driven back along the veins of the head, causing extreme congestion thereof and bleeding into the tissues of the eyeball, ear, mouth, nose, and jaws. A brief note of the influence of atmospheric conditions on the uncovered skin of airmen is contributed by H. S. Schrötter. Dr. Jerusalem describes, with the aid of photographs, typical injuries of the skin and soft parts as the result of trauma due to physical agents. Dr. Wirth considers the skin affections of domestic animals and gives a full account, with illustrations, of the effects produced by pressure of the harness on the skin of horses.

The remaining three papers do not deal directly with injuries due to physical causes. Dr. Fabry gives an account of tuberculosis of the skin with special reference to its occurrence in different occupations; some illustrations are included. Dr. Ravogli deals, in his illustrated article, with sores of the feet and their bearing upon occupation. He finds sores of the leg most common in ordinary laborers (men) and domestic servants (women). An abstract of the laws and decrees of different countries to protect industrial workers is due to Dr. Brezina.

This volume is excellently produced. It contains 128 illustrations in the text, 9 colored plates, and full lists of the bibliography of the subjects under discussion. It constitutes an important work of reference for all engaged in the study of industrial hygiene and of the recent advances in its researches. —*J. A. Campbell.*

INTERNATIONAL CLINICS. Volume II. Thirty-Fourth Series, 1924. Cloth. Pp. 295 and index. Philadelphia and London: J. B. Lippincott Company, 1924.

This volume of International Clinics contains several articles with titles attractive to the industrial physician. There is first of all a symposium on physiotherapy, which begins with a description of physiotherapy technic for intranasal diseases. Anything promising abortive, palliative, or curative treatment for acute and chronic disease in the upper respiratory tract is of interest to industrial medicine. Brooke describes methods evolved in his own clinic and details the apparatus necessary for such work. His results are apparently favorable, but one cannot fail to be struck by the fact that where cases are given they illustrate only the successful possibilities

of the proposed measures. The industrial physician who turns to Brooke's method for relief against one of the great causes of lost time will do so with the realization that he heeds the behest of an enthusiastic advocate and not the substantial representations of a judge.

The second paper, by Dr. Mary Snow, on mechanical vibration in cardiovascular conditions is a further example of the intoxication which so often seizes the physiotherapy practitioner. The paper recounts successful instances of reduction and increase in blood pressure by vibratory treatment applied over the upper dorsal and cervical vertebrae. It is also declared to be possible to improve cardiac tone by appropriate treatments of similar nature. One finds introducing these clinical matters such statements as: "As a test of myocardial insufficiency, register the pulse-rate with the patient lying or sitting, and compare results. Normally it should be higher sitting, from 4 to 6 beats. If found reversed, myocardial insufficiency exists." There is an ingenuous assurance about this which is repeated in other parts of the paper and which does not inspire confidence.

The two remaining papers in the physiotherapy section deal with X-rays and radium and consist of a summary of the physiology and pathology of X-ray action and a readable account of radium treatment.

In the surgical section of the volume there is an exceedingly scholarly and practical article on osteomyelitis by Ashhurst.—*Cecil K. Drinker.*

THE NERVOUS PATIENT. By *Millais Culpin*, M.D., F.R.C.S. Pp. viii, 305. London: H. K. Lewis, Ltd., 1924.

The common sense general practitioner has taken the same attitude toward psycho-analysis as he has preserved toward hypnotism—like Gallio he has cared for none of those things; he has been content to wait and watch. Nevertheless, particularly if his work lies among the industrial population, he is ever more frequently, as the complexity of civilized life increases, encountering the nervous patient whose physical condition is clearly accentuated by some subterranean influence, as for example by the receipt, present or prospective, of a compensation allowance. War-time experience, particularly with regard to cases of disordered action of the heart and shell shock, taught much of the importance of "nerves" among men; he is coming to wonder whether the much spoken of methods of treatment may not be immediately applicable to his own daily work; but he fears the one-sided dicta of experts. Surgeons, however, are essentially materialists; hence when one sets out to discuss the whole matter, the ordinary man will be inclined to listen. The author of this book, M. Culpin, is a surgeon who has become convinced that there are more things in the realm of ill-being than can be covered by material treatment. He sets out the position, grouping clearly the various manifestations and showing how frequently suppression and repression may finally result in physical disorder.

The book gains from an absence of abstruse manufactured terminology which all too frequently pervades works dealing with psychology and entirely obscures for the man in the street the meaning of the text. Occu-

pational neuroses, such as writers' cramp, telegraphers' cramp, railway shock, and other postaccident conditions are discussed and instances are quoted of the presence of anxiety symptoms which, although not conspicuous, were useful as indicating the pathologic state of which the occupational disorder was only part. The warning is issued that reticence concerning mental symptoms so often causes the diagnosis to be overlooked. "The patient who comes with the definite complaint of an occupational cramp falls into one or other subgroup of the minor psychoses; in my experience the cramp subject is more often of the anxiety than the hysterical type, and sometimes is definitely obsessional."

Miners' nystagmus is fairly claimed to be an occupational neurosis, and evidence in favor is drawn from the recent reports issued by the Medical Research Council. This condition, as well as wider issues with regard to eye symptoms, is discussed in a special chapter by an ophthalmic expert, W. S. Inman, whose views with regard to the relationship of eyestrain to nervous symptoms, such as headache, are the reverse of orthodox but are not for that reason false. Some of the cases quoted are indeed illuminating and go far to justify his conclusions that no definite relationship exists between severity of symptoms and the degree of refractive error; and that wrong glasses may cure, while correct glasses may fail.

The industrial physician will find much of value and interest in this volume and as he reads must recognize again and again the similarity between cases quoted and his own daily experience. He, far more than the specialist who seeks for the textbook syndrome and then "makes a diagnosis," knows that mind governs matter and that at the back of the syndrome may lurk *res angusta domi;* and he will welcome an exposition which sets forth plainly much that he has himself learned instinctively through daily and intimate contact with humanity and its troubles.—*E. L. Collis.*

LIGHTING IN RELATION TO PUBLIC HEALTH. By *Janet Howell Clark*, Ph.D., Associate Professor of Physiological Hygiene in the School of Hygiene and Public Health of the Johns Hopkins University. Cloth. Pp. 185 with illustrations and index. Baltimore: Williams & Wilkins Company, 1924.

Professor Clark's recent book provides the public health officer with a textbook on illumination. Only the necessary physical principles are discussed and the treatment is entirely from the point of view of public health. The practical applications of the subject are given in sufficient detail to make the book of real value in field work.

Standards of illumination for various conditions of work, designs for factories, schools, homes, and street lighting, glare, and the best conditions for visual efficiency are the principal topics. In addition to the engineering aspects of the subject, there are three chapters on occupational eye diseases and pathologic effects of radiant energy on the eye, thus combining in one volume the results of modern scientific research in two fields which are usually treated as entirely separate. The utility and convenience to the public health officer of such an arrangement are obvious.—*C. A. Mackay.*